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Health and Environmental Effects Profile for Anthracene

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16. ABSTRACT

The Health and Environmental Effects Profile for anthracene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine a Reference Dose (RfD) or a carcinogenic potency factor for anthracene.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ANTHRACENE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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EXECUTIVE SUMMARY

Anthracene is a colorless solid at ambient temperatures. It is soluble in a variety of organic solvents including ethanol, methanol, benzene, toluene and carbon disulfide (Windholz, 1983), but it is almost insoluble in water (Pearlman et al., 1984). The solubility of anthracene in water decreases slightly with the increase in salt content, but decreases greatly with the lowering of water temperature (Whitehouse, 1984). This compound is susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972). The commercial production of anthracene in the United States is believed to have stopped since 1982 (IARC, 1983), although its U.S. production was >2-20 million pounds in 1977 (U.S. EPA, 1977). In 1983, 30,458 pounds of anthracene was imported into the United States (USITC, 1984). Anthracene is produced commercially from anthracene oil, a coal tar fraction boiling in the range of 270-360°C (IARC, 1983; Hawley, 1981). It is used primarily in the production of dye. Also, small amounts of anthracene are used as a component of smoke screens, as scintillation counter crystals, and in organic semiconductor research (IARC, 1983; Hawley, 1981).

The fate and transport of anthracene in aquatic media has received much more attention than in any other media, because a multicompartment modeling program conducted by Mackay et al. (1985) estimated that >95% of environmental anthracene will reside in the aquatic compartment. The fate and transport of anthracene in surface waters will depend on the nature of the water. In most waters, the loss of anthracene is mainly due to photolysis and biodegradation (Mackay et al., 1985); however, in a very shallow, fast-flowing clear water, volatilization and photolysis will play dominant roles in determining the fate of anthracene (Southworth, 1979). In deep, slow

flowing and muddy waters, microbial degradation and adsorption may account for the major losses of anthracene from water (Southworth, 1979). Therefore, the half-life of anthracene in natural surface waters will depend on the nature of the water bodies. In very shallow, fast-flowing and clear water, its half-life may be ~1 hour (Southworth, 1979; Herbes et al., 1980). On the other hand, the half-life may be as high as 29 days in a deep eutrophic pond (Zepp, 1980).

In air, anthracene is expected to be present both in the vapor and the particle-sorbed state. Over 78% of atmospheric anthracene may be present in the vapor state (Thrane and Mikalsen, 1981). Both chemical processes including O_3 and OH radical and photochemical reaction will degrade atmospheric anthracene (Niessner et al., 1985; Atkinson, 1985; Korfmacher et al., 1980; Behymer and Hites, 1985). The degradation of vapor phase atmospheric anthracene is expected to be faster than particle-sorbed anthracene (Santodonato et al., 1981). The atmospheric half-life of anthracene may vary from hours to days (Atkinson, 1985; Korfmacher et al., 1980; Behymer and Hites, 1985; Lunde and Bjoerseth, 1977). The long range transport of anthracene observed by Lunde and Bjoerseth (1977) indicates that particle-sorbed anthracene may have a half-life of the order of days.

The fate and transport of anthracene in soils is not well studied. Both biodegradation and abiotic processes will degrade anthracene in soils (Bossert et al., 1984). The half-life of anthracene in soil may be ~1 month (Bossert et al., 1984). Anthracene may not leach from most soils because of its high K_{oc} value; however, it may leach through soils that have attained the breakthrough capacity for anthracene sorption (Piet and Morra, 1979).

Anthracene is ubiquitous in the aquatic environment. Anthracene at a concentration range of <13-105 ng/ℓ was reported in the effluent from a sewage treatment plant in Norway (Kveseth et al., 1982). It was also reported in the concentration range of <0.03-0.84 µg/ℓ in the effluent from coal oven plants (Walters and Luthy, 1984; Griest, 1980). It has been reported to be present in several surface waters (Hites, 1979; Dewalle and Chian, 1978; Staples et al., 1985). A maximum concentration of 12.1 µg/ℓ was reported in the water from Tennessee River in Calvert City, KY (Goodley and Gordon, 1976). In general, anthracene occurs in ambient waters at a frequency of 4% and at a median concentration of <10 µg/ℓ (Staples et al., 1985). Anthracene has been reported to be present in groundwater from a few contaminated sites. Bedient et al. (1984) reported the detection of up to 168.6 µg/ℓ of anthracene in groundwater from a creosote waste site in Conroe, TX. The detection of anthracene in drinking waters throughout the world have been reported (Williams et al., 1982; Kveseth et al., 1982; Shinohara et al., 1981; Piet and Morra, 1979). The highest concentration of combined anthracene/phenanthrene at 1269 ng/ℓ was reported in drinking water in Sault Ste. Marie (Williams et al., 1982). Finished waters from 13 different locations throughout the United States, however, failed to show the presence of any anthracene (Sorrell et al., 1980).

The concentrations of anthracene in ambient air in some cities in the United States and around the world have been reported. The mean concentration of anthracene in the air of Los Angeles during 1981-82 was reported to be 0.54 ng/m³ (Grosjean, 1983), whereas its mean concentration in Osaka, Japan, during the same period was reported to be 0.32 ng/m³ (Matsumoto and Kashimoto, 1985). It has been demonstrated by Grosjean (1983) that the

atmospheric anthracene concentrations show both seasonal and diurnal variations, with the anthracene concentration higher in winter and during the night. Although suitable data were not available on anthracene for comparison, Grosjean (1983) estimated that the levels of other polynuclear aromatic hydrocarbons in Los Angeles air did not change significantly during the last decade. Based on the assumptions that the mean level of anthracene in U.S. air is 0.54 ng/m³ (same as concentration in Los Angeles) and that an adult individual inhales 20 m³ air/day, the average daily intake of anthracene from inhalation is 11 ng.

Anthracene has been reported to be present in smoked foods, liquid smoke, charcoal-broiled steaks and edible aquatic organisms collected from certain contaminated waters (Fazio and Howard, 1983; Galloway et al., 1983). For example, the concentrations of anthracene in charcoal-broiled steaks and barbecued ribs were reported to be 4.5 and 7.1 µg/kg, respectively (Fazio and Howard, 1983). The U.S. National Mussel watch program collected mussels from >100 sites on the East, West and Gulf coasts (Galloway et al., 1983). The concentration of anthracene in these mussel composites was 7.9-32 µg/kg (Galloway et al., 1983). Until data on the level of this compound in food composites used by an individual in the United States are available, it is not possible to estimate the human intake of anthracene through consumption of foods in the United States.

There is relatively little information concerning the toxicity of anthracene to aquatic organisms. Acutely toxic concentrations range from 1.9 µg/ℓ for Daphnia pulex (Allred and Giesy, 1985; Oris et al., 1984) to 3030 µg/ℓ for Daphnia magna (Bobra et al., 1983). Some of this variability may be explained by the fact that anthracene toxicity is affected by lighting conditions, with toxicity increased under natural

sunlight and ultraviolet radiation rather than fluorescent lights (Allred and Giesy, 1985; Bowling et al., 1983; Kagan et al., 1985). Reported toxic concentrations for aquatic plants were also highly variable. Hutchinson et al. (1980) reported that photosynthesis was inhibited in Chlamydomonas angulosa at 239 µg/ℓ, while Biddings (1979) found that Selenastrum capricornutum was unaffected by a 100% saturated solution. Reported BCF values ranged from 47-132 for Chironomus riparius (Gerould et al., 1983) to 16,600 for Pontoporlia hoyi (Landrum, 1982). These species also represent the extremes in the ability to metabolize and eliminate anthracene.

Limited information is available regarding the pharmacokinetic profile of anthracene. Gastrointestinal absorption may be poor, as 53-84% of anthracene administered by diet or stomach tube was eliminated in the feces by rats in 2-3 days (Chang, 1943); however, urinary and biliary excretion were not measured. Radioactivity from single intratracheal instillations of ^{14}C-anthracene was cleared from the lungs in a biphasic manner with half-times of 0.1 hours (99.7% of dose) and 25.6 hours (0.3% of dose) (Bond et al., 1985). The distribution of anthracene to tissues does not appear to have been investigated. Metabolites resulting from epoxidation at the 1,2-bond or oxidation at the 9- and 10-positions have been identified in in vivo and in vitro studies with rats (Sims, 1964; Akhtar et al., 1979). Trans-1,2-dihydroxy-1,2-dihydroanthracene and sulfate and glucuronide conjugates consistent with the formation of anthracene-1,2-oxide appear to be the major products. Orally administered anthracene appears to be eliminated by rats primarily (53-84%) in the feces (Chang, 1943). Metabolites of orally administered anthracene have been detected in the urine of rats (Sims, 1964).

Administration of diets that supplied a total dose of 4.5 g/rat of anthracene over 78 weeks produced tumors in 2/28 rats (a liver sarcoma and a uterine adenocarcinoma) that were observed for life (Schmahl, 1955). A control group was not used and the tumors were not ascribed to treatment. A single intrapulmonary injection of 0.5 mg anthracene in beeswax-tricaprylin mixture did not induce local neoplastic responses in rats after 4-55 weeks of observation (Stanton et al., 1972).

Twice or thrice weekly skin applications of anthracene for life did not produce local tumors in mice (Bachmann et al., 1937; Wynder and Hoffmann, 1959; Miescher, 1942), but contradictory results were obtained when anthracene was applied to mouse skin with concurrent or directly subsequent ultraviolet irradiation (Heller, 1950; Forbes et al., 1976). Mouse skin initiation-promotion assays using croton oil (Salaman and Roe, 1956) or TPA (Scribner, 1973) as the promoter did not indicate a tumor initiating effect of anthracene. Weekly subcutaneous injections of anthracene in rats for 6 weeks to life (Pollia, 1941; Schmahl, 1955; Boyland and Burrows, 1935), weekly intraperitoneal injections in rats for 33 weeks (Schmahl, 1955) or brain or eye implants in rabbits for 4.5 years (Russell, 1947) did not produce local tumors, but these findings should be regarded as inconclusive because of inadequacies in experimental design. Taken together, the available human and animal data for carcinogenicity are inadequate, EPA Group D, to make an assessment of human carcinogenic activity.

Anthracene has been tested in numerous mutagenicity and other short-term assays with primarily negative results (IARC, 1983; Langenbach et al., 1983; Lubet et al., 1983; Ved Brat et al., 1983; Mamber et al., 1984; Quillardet

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et al., 1985). These include DNA damage, mutation, cytogenicity and transformation assays with bacteria, yeast and mammalian cells *in vitro* and *in vivo*. Exogenous metabolic activation systems were used in most of the *in vitro* assays.

Pertinent data regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of anthracene could not be located in the available literature as cited in the Appendix.

Data were insufficient to derive an RfD, q_1^*, RQ or F factor, and thus, a hazard ranking is precluded.

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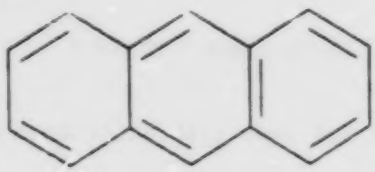
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
CAS	Chemical Abstract Service
DMSO	Dimethyl sulfoxide
DNA	Deoxyribonucleic acid
EC_{50}	Concentration effective to 50% of recipients
GC	Gas chromatography
HGPRT	Hypoxanthine-guanine phosphoribosyl transferase
K_{oc}	Soil sorption coefficient standardized with respect to organic carbon
K_{ow}	Octanol/water partition coefficient
LD_{50}	Dose lethal to 50% of recipients
MED	Minimum effective dose
PAH	Polynuclear aromatic hydrocarbon
ppb	Parts per billion
ppm	Parts per million
RQ	Reportable quantity
TLV	Threshold limit value
TPA	Terephthalic acid
TWA	Time-weighted average

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

The chemical commonly known as anthracene is also known by the synonyms paranaphthalene, green oil and tetra olive NZG (U.S. EPA, 1986a). The structure, empirical formula, molecular weight and CAS Registry number for this chemical are as follows:



Empirical formula: $C_{14}H_{10}$

Molecular weight: 178.22

CAS Registry number: 120-12-7

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Anthracene is a colorless crystalline compound at ambient temperatures. It is almost insoluble in water but is soluble in a variety of organic solvents including ethanol, methanol, benzene, toluene and carbon disulfide (Windholz, 1983). Some of the relevant physical properties of anthracene are listed below.

Melting point:	216°C	Santodonato et al., 1981
Boiling point:	340°C	Santodonato et al., 1981
Density at 27/4°C:	1.25	Windholz, 1983
Water solubility:		
Distilled water at 25°C:	0.041-0.080 mg/ℓ	Pearlman et al., 1984
	0.066 mg/ℓ (average)	Pearlman et al., 1984
at 25.3°C:	0.044 mg/ℓ	Whitehouse, 1984
at 4.6°C:	0.010 mg/ℓ	Whitehouse, 1984
Distilled water with 36.5% salinity at 25.3°C:	0.032 mg/ℓ	Whitehouse, 1984

Log K_{ow}:	4.45-4.63	Ruepert et al., 1985; Readman et al., 1982; Ogata et al., 1984; Yalkowsky and Valvani, 1979
Vapor pressure:	7.65×10^{-6} mm Hg at 20°C	Grayson and Fosbraey, 1982
	5.65×10^{-6} to 8.48×10^{-6} mm Hg at 25°C	Sears and Hopke, 1949; McEachern and Sandoval, 1973; Etzweiler et al., 1984
Henry's Law constant:	3.64×10^{-5} atmos·m³/mol	Webster et al., 1985

It can be concluded from these data that the solubility of anthracene in water decreases slightly with the increase of salt content, but the solubility is greatly dependent on the temperature of the water. Chemically, polycyclic aromatic hydrocarbons are reasonably reactive. They can undergo substitution and addition reactions. In addition, these compounds are susceptible to oxidation by ozone, peroxides and other oxidants (NAS, 1972).

1.3. PRODUCTION DATA

According to the nonconfidential portion of the TSCA production file (U.S. EPA, 1977), Koppers Co., Inc., reported production of a total of 2-20 million pounds of anthracene at two sites in 1977, and DuPont Co. produced <1000 pounds of the chemical during the same year. Allied Chemical also reported production of anthracene in 1977 but did not report the production volume. The commercial production of anthracene in the United States is believed to have stopped since 1982 (IARC, 1983). In 1983, the amount of anthracene imported to the United States was 30,458 pounds (USITC, 1984).

Anthracene is commercially produced from anthracene oil, a coal-tar fraction boiling in the range of 270-360°C. Crude anthracene is obtained from anthracene oil either by crystallization or by distillation of the oil.

The crude product is purified by washing with appropriate solvents followed by sublimation. Pure crystals of anthracene are obtained by zone refining of solid anthracene. Commercial anthracene is ~90-95% pure by weight. The major impurities in commercial anthracene are: carbazole (3% max), pyridine (0.2% max), iron (0.03% max), phenanthrene and chrysene (IARC, 1983; Hawley, 1981).

1.4. USE DATA

Anthracene is used primarily as an intermediate in dye production; it is no longer used in the United States for the commercial production of anthraquinone for dye usage. Anthracene is also used as a component of smoke screens, as scintillation counter crystals, and in organic semiconductor research (IARC, 1983; Hawley, 1981).

1.5. SUMMARY

Anthracene is a colorless solid at ambient temperatures. It is soluble in a variety of organic solvents including ethanol, methanol, benzene, toluene and carbon disulfide (Windholz, 1983), but it is almost insoluble in water (Pearlman et al., 1984). The solubility of anthracene in water decreases slightly with the increase in salt content, but decreases greatly with the lowering of water temperature (Whitehouse, 1984). This compound is susceptible to oxidation by ozore, peroxides and other oxidants (NAS, 1972). The commercial production of anthracene in the United States is believed to have stopped since 1982 (IARC, 1983), although its U.S. production was >2-20 million pounds in 1977 (U.S. EPA, 1977). In 1983, 30,458 pounds of anthracene was imported into the United States (USITC, 1984). Anthracene is produced commercially from anthracene oil, a coal tar fraction boiling in the range of 270-360°C (IARC, 1983; Hawley, 1981). It is used primarily in

the production of dye. Also, small amounts of anthracene are used as a component of smoke screens, as scintillation counter crystals, and in organic semiconductor research (IARC, 1983; Hawley, 1981).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Photodegradation. The photolysis of anthracene in water was studied by Zepp and Schlotzhauer (1979). It was observed by these authors that photoreactions are more efficient in water than in hydrocarbon solvents. Besides the intensity of solar radiation, which depends on the season of the year, the time of day and the latitude of the site, the photoreaction rate also depends on the depth and turbidity of the water bodies. In turbid waters, the photodegradation rate is slower because of light attenuation, which is due to scattering, and the partitioning of anthracene between water and sediment. The portion of anthracene that may be present in the sediment as a result of adsorption will not photodegrade because of the inability of light to reach the bottom. Zepp and Schlotzhauer (1979) computed the near-surface half-life for direct photochemical transformation of anthracene at 40°N latitude by mid-day, midsummer sun to be ~1 hour. Because of the attenuation of solar radiation, the average half-life in the top 35 m of water from the middle of the Gulf of Mexico was computed to be 6 hours. Since the coastal water near Tampa, FL, was more turbid, the computed average half-life was >1 day (Zepp and Schlotzhauer, 1979). Besides direct photoreaction, the indirect photooxidation of anthracene mediated by singlet oxygen was also studied by Zepp and Schlotzhauer (1979). The half-life for this photosensitized oxidation of anthracene was computed to be 200 hours. Therefore, photosensitized oxidation in natural waters was concluded to be too slow to compete with direct photolysis.

The photolysis of anthracene in distilled water was also reported by Southworth (1979). Exposure of anthracene solution (~10 ppb) to midsummer mid-day sunlight at 35°N latitude rapidly degraded anthracene with a

half-life of 35 minutes. Under average winter and summer solar conditions at the same latitude, this corresponds to photolytic half-lives of 4.8 hours and 1.6 hours, respectively. In most natural waters, the absorption of light by dissolved and suspended matter and the depth of water would act to reduce photolysis rates considerably. Thus, the photolytic half-lives of anthracene in natural waters with decadic light absorption coefficients of 0.020 and 0.100, which is due to an increase in turbidity, were estimated to increase by factors of 4 and 19, respectively, compared with distilled water photolysis (Southworth, 1979).

2.1.2. Chemical Reactions. The rate constant for the reaction of anthracene with singlet oxygen ($^1\Delta_g$) in benzene at 25°C was reported to be 0.15×10^6 M^{-1} sec^{-1} (Stevens et al., 1974). Assuming the rate constant in aquatic media to be similar and the concentration of singlet oxygen in natural water to be 10^{-12} M (Mill et al., 1982), the half-life for this reaction can be estimated to be >50 days. Therefore, this reaction is not likely to be environmentally significant. The reaction of anthracene with ozone, which may be environmentally significant if ozonation is used as a method of disinfecting drinking water, was studied by Kuo and Barnes (1985). The reaction between anthracene and ozone in aqueous solution was found to be extremely fast and the estimated half-life was less than a few milliseconds; however, the products of the ozonation were not reported by the authors.

2.1.3. Biodegradation. The biodegradability of anthracene has been studied with pure cultures of microorganisms, mixed microorganisms and natural water. Several pure cultures of microorganisms including Flavobacterium sp., Pseudomonas aeruginosa, Pseudomonas putida, Nocardia sp. (Fuhs, 1961; McKenna, 1977), Beijerinckia sp. (Gibson, 1977), Vibrio sp.,

Bacillus sp., Candida sp., Arthrobacter sp. (Bhosle and Mavinkurve, 1980) and Alealigenes faecalis (Kiyohara et al., 1982) were shown to biodegrade anthracene. Although these pure culture studies are far removed from the natural conditions for biodegradation, they are useful in establishing biodegradation pathways of chemicals. The proposed pathway for bacterial catabolism of anthracene is as follows (Cerniglia, 1981; Ribbons and Eaton, 1982; McKenna and Kallio, 1965; Subramanian et al., 1978; Van der Linden and Thijsse, 1965):

anthracene → 1,2-dihydroxyanthracene → 2-hydroxy-3-naphthaldehyde → 2-hydroxy-3-naphthoic acid → 2,3-dihydroxynaphthalene → salicylic acid

The biodegradability of anthracene with mixed microorganisms was studied by several authors. Thom and Agg (1975) reported that anthracene is biodegradable in sewage treatment plants provided suitable acclimatization can be achieved. With settled domestic wastewater as microbial inoculum and a static-culture flask-screening procedure, 43% of anthracene was found to be biodegradable in 7 days at an initial concentration of 5 ppm. After 7 days of acclimatization, the same solution showed 70% degradation in 7 days. The corresponding degradation was only 26 and 30% at an initial anthracene concentration of 10 ppm (Tabak et al., 1981). Lutin et al. (1965) used activated sludge from three municipal treatment plants as microbial inoculum and the Warburg method as a means for estimating the rate of biooxidation. Anthracene was reported to be appreciably resistant to biodegradation with activated sludge from two municipal plants, and degradable with the third activated sludge. Freitag et al. (1985) reported only 0.3% CO_2 formation (relative to applied dose) on incubation of anthracene for 5 days with activated sludge.

The biodegradability of anthracene with natural sediments (Gardner et al., 197?; Bauer and Capone, 1985; Schwall and Herbes, 1979; Herbes, 1981; Herbes and Schwall, 1978; Lee and Ryan, 1983) and natural estuarine waters (Lee and Ryan, 1983; Lee, 1977) has been studied by several authors. Several conclusions can be reached from these studies. The biodegradation of anthracene in aquatic media is controlled by the temperature, oxygen content and acclimatization/nonacclimatization of the microorganisms. Higher biodegradation rates were observed at 30°C than at 20 and 10°C (Bauer and Capone, 1985). The biodegradation process was found to be aerobic and higher oxygen concentration up to a certain optimum value tended to increase the oxidation rates (Bauer and Capone, 1985). Similarly, the biodegradation rates were reported to be faster with acclimatized microorganisms (Bauer and Capone, 1985; Herbes and Schwall, 1978; Lee and Ryan, 1983; Lee, 1977). The incubation of anthracene with intertidal sediment slurries for a reasonable period of time (~1 month) not only produces the mineralization product CO_2 but also produces intermediate metabolites. A large portion of the initial material or its intermediate metabolites (which could not be identified because ^{14}C counting of the combustion products of residue was used as the method of quantification) remained cellular bound (Bauer and Capone, 1985; Schwall and Herbes, 1979). For example, incubation of anthracene with petroleum-contaminated sediments for 5 days at 12°C produced ~42% of extractable unaltered anthracene, 4% of CO_2 and 4% of polar metabolites; ~30% remained cellular bound (Schwall and Herbes, 1979). The residual 20% of the original anthracene remained unaccounted for. The half-lives of anthracene in waters and sediments are generally expressed as the complete mineralization half-life and the half-life for the disappearance of initial

anthracene. Although the former method correctly predicts the mineralization half-life, it does not reflect the loss of anthracene that is due to formation of intermediate metabolites. Therefore, it always overestimates the half-life for biodegradation of anthracene. The latter method, on the other hand, may underestimate the biodegradation half-life because of the inability of the quantification method to determine the amount of starting material in the cellular bound part and to obtain a complete material balance for the starting material. The mineralization half-life of anthracene has been reported to be 57-210 days in unacclimatized sediments and 5-7 days in oil treated sediments (Lee and Ryan, 1983). The mineralization half-life of anthracene was also reported to be ~200 days in oil-treated water and ~20-fold higher in uncontaminated water (Lee, 1977). The overall biotransformation (both CO_2 and intermediate metabolite formation) half-life of anthracene in petroleum-contaminated sediment was reported to be ~12 days (Herbes and Schwall, 1978). In pristine sediments, the overall biotransformation half-life was ~10-fold higher (Herbes and Schwall, 1978). The overall biotransformation half-life of anthracene in sediments contaminated with a coal-coking wastewater was ~2 days. The anthracene transformation rate was ~20 times lower in the water (Herbes, 1981).

2.1.4. Volatilization. The rates of volatilization of anthracene from bodies of water were studied by several investigators. Besides the water temperatures, the rates of volatilization are dependent on the depth of the water, the current of the flowing water, the wind velocity above the water and the nature and amount of suspended solids present in the water (Southworth, 1979). Decreases in water depths, increases in current and wind velocity and decreases in absorption onto suspended particles in water are expected to increase the volatilization rates. None of the available

estimation methods for the determination of evaporative half-life of anthracene in water bodies, however, incorporates the effect of sorption on volatilization. The estimated evaporative rate constants, half-lives and percent loss that is due to evaporation from different water bodies as estimated from gas and liquid phase exchange constants and Henry's Law constant are shown in Table 2-1.

2.1.5. Adsorption. The adsorption of a compound to suspended particulate matter and sediments can be predicted from its K_{oc} value, which is ~26,000 for anthracene (Karickhoff et al., 1979). This is indicative of the possibility of strong adsorption of anthracene onto suspended particles and sediments in water. Several investigators have estimated, however, that the removal rate of anthracene from water that is due to adsorption is small compared with other removal processes. Depending on the nature of the water body, the percent of anthracene removal from water may vary from negligible to 18%. The percent removal by adsorption was estimated to be maximum in deep, slow moving, muddy water bodies (Southworth, 1979). Depending on the characteristics of water bodies, Herbes et al. (1980) also estimated that between 2.8 and 19% of anthracene removal will take place by adsorption. The EXAMS computer modeling of a 2 m deep eutrophic pond and 3 m deep river with an input K_{oc} value of 26,000 estimated that ~3.3 and 0.4%, respectively, of anthracene will be removed by sedimentation (Zepp, 1980).

2.1.6. Bioconcentration. The bioconcentrations of chemicals in aquatic organisms are dependent on the overall uptake and elimination kinetics. Unless an equilibrium is attained between these two opposing processes, the bioconcentration factors are expected to vary. With the possible exception of the results of Freitag et al. (1982), BCFs for anthracene in different

TABLE 2-1

Removal of Anthracene by Volatilization from Water Bodies at 25°C*

Water Velocity (m/sec)	Wind Velocity (m/sec)	Depth	Half-Life	% Loss of Total Due to Volatilization
0.1	4	5 m	14 days	2-3
0.1	0.1	5 m	29 days	3
1.0	4.0	1 m	16.5 hours	21
0.5	1.0	1 m	62 hours	7.7
1	4	25 cm	4 hours	35

*Source: Southworth, 1979; Herbes et al., 1980

aquatic organisms given in Table 2-2 were obtained under apparent equilibrium conditions. Additional data regarding BCFs are presented in Chapter 6. McCarthy (1983) pointed out that the uptake of PAHs including anthracene is greatly reduced with sorbed chemical compared with its uptake from the dissolved state. Therefore, natural waters containing anthracene in the suspended organic matter in the sorbed state are likely to show BCFs that differ from those obtained in laboratory experiments with particulate-free water. In the case of anthracene, the differences may not be pronounced because the removal rate of anthracene from water that is due to adsorption is small compared with other removal processes (see Section 2.1.5.). It can be concluded from Table 2-2 that anthracene moderately bioconcentrates in aquatic organisms.

2.1.7. Overall Removal Processes. While the rate of uptake by fish is faster than the rate of uptake by particulates, a high concentration of suspended particles in water may offset this rate difference. Thus, under conditions of high concentration of suspended particles, BCF values may be lower. The removal of anthracene from aquatic media will depend on the nature of the aquatic media. The predicted contributions of major processes in the removal of anthracene from different aquatic media are given in Table 2-3, which indicates that, in most water bodies, photolysis and microbial degradation will play important roles in the loss of anthracene. There is a large difference, however, in the predicted half-life of anthracene between the Southworth (1979) investigation and the Zepp (1980) investigation. The former's values are probably underestimated because they were derived without full consideration of effects of sorption on volatilization, photolysis and microbial degradation. The values derived by Zepp (1980), using similar input parameters but using EXAMS computer model, may not be accurate. In a

TABLE 2-2

BCFs for Anthracene in Different Aquatic Organisms

Species	BCF	Reference
Alga (*Oedogonium cardiacum*)	670	Lu et al., 1978
Snail (*Physa* sp.)	2714	Lu et al., 1978
Fish (*Gambusia affinis*)	1029	Lu et al., 1978
Golden orfe (*Leuciscus idus melanotus*)	910	Freitag et al., 1982
Goldfish (*Carassius auratus*)	162	Ogata et al., 1984
Zooplanker (*Daphnia pulex*)	917	Southworth et al., 1978

TABLE 2-3

Contributions of Major Processes in Removal of Anthracene from Water at 25°C During Midsummer

Water Characteristics	Process	% Contribution	Overall Half-life (hours)	Reference
Deep, slow and somewhat turbid	volatilization	3	10.5	Southworth, 1979
	adsorption	0		
	photolysis	5		
	microbial degradation	91		
Deep, slow and muddy	volatilization	3	21.6	Southworth, 1979
	adsorption	18		
	photolysis	0		
	microbial degradation	79		
Deep, slow and clear	volatilization	2	8.5	Southworth, 1979
	adsorption	1		
	photolysis	22		
	microbial degradation	74		
Shallow, fast and clear	volatilization	21	3.5	Southworth, 1979
	adsorption	4		
	photolysis	44		
	microbial degradation	31		
Very shallow, fast and clear	volatilization	35	1.4	Southworth, 1979
	adsorption	6		
	photolysis	47		
	microbial degradation	12		
2 m deep, eutrophic pond	volatilization	2.4	696	Zepp, 1980
	waterborne export	2.9		
	photolysis	89		
	microbial degradation	6.1		
3 m deep, river	volatilization	4.7	62	Zepp, 1980
	waterborne export	38		
	photolysis	56		
	microbial degradation	0.5		
Shallow, rapidly flowing clear stream	volatilization	7.5	1	Herbes et al., 1980
	adsorption	20.4		
	photolysis	71.4		
	microbial degradation	0.7		

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eutrophic pond where algae growth will prevent light penetration into the interior of water, ~90% photodegradation of anthracene appears to be an overestimate.

2.2. AIR

The fate and transport of anthracene in the atmospheric media has received much less attention than in aquatic media, because a multicompartment (six) modeling program conducted by Mackay et al. (1985) estimated that >95% of environmental anthracene will reside in water and <1.4 in atmospheric media.

The reactivity of atmospheric anthracene will be largely dependent on the state in which it exists in the atmosphere. The reactivity of vapor phase anthracene is expected to be different from its reactivity in the adsorbed state (Santodonato et al., 1981). Considering the vapor pressure of anthracene (see Section 1.2.), the compound is expected to exist both in the vapor and the particle-sorbed phase in the atmosphere (Eisenreich et al., 1981). The experimental results of Thrane and Mikalsen (1981) indicate that from 78-98% of anthracene collected from the atmosphere exists in the vapor phase. The reactivity of anthracene with atmospheric oxidants including OH radical, O_3 and NO_2 has not been studied comprehensively. The heterogeneous reaction of gas phase O_3 and NO_2 with anthracene coated on sodium chloride was studied by Niessner et al. (1985), who reported that the heterogeneous reaction of anthracene was negligible with NO_2 but reaction with O_3 was significant. Both anthrone and anthraquinone were reported to be the products of this reaction. Although this experiment points out the possibility of O_3 reaction with anthracene in the atmosphere, in the absence of rate data it cannot be used for predicting quantitatively the fate of anthracene in the ambient air that is due to this reaction. The

rate constant for the reaction of gaseous anthracene with hydroxyl radicals at 52°C is reported to be 112×10^{-12} cm³/molecule-sec (Atkinson, 1985). If the concentration of OH radical in the atmosphere is assumed to be 10^6 radicals/cm³ (U.S. EPA, 1980a), the half-life of this reaction at 52°C is ~2 hours. In the troposphere where the average temperature is <52°C and part of the anthracene will exist in the particle-sorbed phase, the half-life may be longer than the value estimated at 52°C.

Based on its fate in aquatic media, photodegradation is expected to be a significant process in the atmosphere as well. The photoreaction of particle-sorbed anthracene was shown to be substrate-dependent (Behymer and Hites, 1985). For example, when anthracene was adsorbed onto silica gel, alumina, fly ash and carbon black and irradiated with light from a medium pressure mercury arc in a rotary photoreactor, the corresponding half-lives were 2.9, 0.5, 48 and 310 hours. Thus, it appears that both fly ash and carbon black stabilize anthracene in the atmosphere and may facilitate their transport from combustion sources. Similar conclusions were reached by Korfmacher et al. (1980), who reported that anthracene adsorbed on fly ash was resistant to photodecomposition. Photoreaction of anthracene that has been dispersed into atmospheric particulate matter was also reported by Fox and Olive (1979). These authors concluded that photodegradation in the atmosphere is a more important fate process than ozonation. When anthracene adsorbed onto atmospheric particulate matter was exposed to bright sunshine for 4 days, ~10% of the compound disappeared. The authors concluded that besides oxidation products (anthrone, anthraquinone), photolysis may also produce polymeric condensed products. Therefore, the following conclusions can be reached regarding the photoreaction of anthracene in the atmosphere: 1) the majority of anthracene that will be present in the atmosphere in the

unadsorbed gas phase will photodegrade by atmospheric sunlight with a half-life of a few hours, as in the case of solution phase anthracene (see Section 2.1.1. and Korfmacher et al., 1980); 2) the relatively small fraction of atmospheric anthracene that will exist in the particle-sorbed state will be more resistant to photodegradation and may degrade by sunlight with a half-life of days.

Atmospheric anthracene can also be removed by physical processes. The removal of atmospheric anthracene in the Great Lakes ecosystem was reported by Eisenreich et al. (1981). Both dry deposition of the vapor and particle-bound anthracene and wet precipitation through rain and snow occurred; however, dry deposition was found to be more important than wet deposition. Ligocki et al. (1985a) concluded that particle scavenging was less important than gas scavenging of atmospheric anthracene. The half-lives for these physical removal mechanisms were not provided by either study; however, the removal of atmospheric anthracene through these physical mechanisms appear to be less significant than its removal through biotic processes. Finally, Lunde and Bjoerseth (1977) reported that the concentration of anthracene that originated from trajectories from Western Europe contained ~4 times more anthracene than samples with trajectories from northern Norway or stationary air from southern Norway. This result suggests that at least a part of atmospheric anthracene is stable enough in the atmosphere to travel long distances. Perhaps the part of anthracene in the atmosphere that existed in the particle-sorbed phase, remained nonreactive towards abiotic processes, and was not removed by physical processes underwent this long distance transport in the atmosphere.

2.3. SOIL

The fate of anthracene in soils is studied even less than its fate in the atmosphere. Its fate in soil can be predicted, however, from the knowledge of its fate in aquatic media. The three processes that are important in the loss of anthracene from aquatic media are photolysis, biodegradation and volatilization. Because of light attenuation and scattering, photolysis cannot be an important process for the loss of anthracene beyond the surficial layer of soils. It has been shown by Bossert et al. (1984) that oily sludge containing anthracene and other polynuclear aromatic compounds when incorporated in a sandy loam soil underwent loss of anthracene. Since multiple applications of sludge to soil were made at a certain interval with an intervening nonapplication period, it is difficult from the data presented to estimate the degradation half-life of anthracene in the soil. On the basis of the decay of the chemical after its first application, the half-life is ~32 days. The authors concluded that biodegradation and abiotic processes accounted for observed decreases in the concentration. The undefined abiotic processes were responsible for <50% of the loss of anthracene from the soil. Some of the loss was speculated to be due to volatilization.

The possibility of leaching of anthracene from soil to groundwater will depend on soil type. The K_{oc} value for anthracene is ~26,000 (Karickhoff et al., 1979). This indicates that anthracene will be adsorbed strongly to soil and the compound may degrade before it reaches groundwater. Filtration of polluted surface water containing anthracene through sandy soil at a residence time of ~100 days did not completely eliminate anthracene in the filtered water (Piet and Morra, 1979). The passage of anthracene through the soil was explained as a breakthrough of the chemical because of the saturation of active sorption sites.

2.4. SUMMARY

The fate and transport of anthracene in aquatic media has received much more attention than in any other media, because a multicompartment modeling program conducted by Mackay et al. (1985) estimated that >95% of environmental anthracene will reside in the aquatic compartment. The fate and transport of anthracene in surface waters will depend on the nature of the water. In most waters, the loss of anthracene is mainly due to photolysis and biodegradation (Mackay et al., 1985); however, in a very shallow, fast-flowing clear water, volatilization and photolysis will play dominant roles in determining the fate of anthracene (Southworth, 1979). In deep, slow flowing and muddy waters, microbial degradation and adsorption may account for the major losses of anthracene from water (Southworth, 1979). Therefore, the half-life of anthracene in natural surface waters will depend on the nature of the water bodies. In very shallow, fast-flowing and clear water, its half-life may be ~1 hour (Southworth, 1979; Herbes et al., 1980). On the other hand, the half-life may be as high as 29 days in a deep eutrophic pond (Zepp, 1980). Anthracene will moderately bioconcentrate in aquatic organisms.

In air, anthracene is expected to be present both in the vapor and the particle-sorbed state. Over 78% of atmospheric anthracene may be present in the vapor state (Thrane and Mikalsen, 1981). Both chemical processes including O_3 and OH radical and photochemical reaction will degrade atmospheric anthracene (Niessner et al., 1985; Atkinson, 1985; Korfmacher et al., 1980; Behymer and Hites, 1985). The degradation of vapor phase atmospheric anthracene is expected to be faster than particle-sorbed anthracene (Santodonato et al., 1981). The atmospheric half-life of anthracene may

vary from hours to days (Atkinson, 1985; Korfmacher et al., 1980; Behymer and Hites, 1985; Lunde and Bjeorseth, 1977). The long range transport of anthracene observed by Lunde and Bjoerseth (1977) indicates that particle-sorbed anthracene may have a half-life of the order of days.

The fate and transport of anthracene in soils is not well studied. Both biodegradation and abiotic processes will degrade anthracene in soils (Bossert et al., 1984). The half-life of anthracene in soil may be ~1 month (Bossert et al., 1984). Anthracene may not leach from most soils because of its high K_{oc} value; however, it may leach through soils that have attained the breakthrough capacity for anthracene sorption (Piet and Morra, 1979).

3. EXPOSURE

3.1. WATER

Anthracene is ubiquitous in the aquatic environment. It has been detected in industrial effluents, in run-off waters, in surface water and sediments, in groundwater, and in drinking water. The industrial effluents that are most likely to contain polynuclear aromatic compounds including anthracene are wastewaters from the synfuel industry, shale oil plants, petroleum processing plants, other industries using coal derived products, wastewater treatment plants and aluminum reduction plants (Bjoerseth et al., 1978; Gammage, 1983; Kveseth et al., 1982); however, anthracene has been reported in effluents from only a few industries. Anthracene at a concentration range of <13-105 ng/ℓ was reported in the effluent from a sewage treatment plant in Norway (Kveseth et al., 1982). Other investigators have reported anthracene at a concentration range of <0.03-0.84 µg/ℓ in the effluent from coke oven plants (Walters and Luthy, 1984; Griest, 1980). The detection of anthracene was also reported in urban run-off waters. Cole et al. (1984) monitored run-off waters from 15 U.S. cities and detected an 8% frequency of anthracene and a concentration range of 1-10 µg/ℓ. Anthracene was detected at trace levels in the water of a small segment of the Delaware River, north of Philadelphia (Hites, 1979). Dewalle and Chian (1978) analyzed water from the Delaware River and its major tributaries and detected anthracene at a concentration of >1 µg/ℓ with a frequency of 3%. Samples of water taken from the Tennessee River in Calvert City, KY, were reported to contain anthracene at a concentration of 12.1 µg/ℓ (Goodley and Gordon, 1976). Since 1980, U.S. EPA collected 776 ambient water samples for the analysis of anthracene (Staples et al., 1985).

Anthracene was detected in 4% of the samples and at a median concentration of <10 µg/ℓ. Anthracene was also reported in surface wastes in England (Fielding et al., 1981) and in Germany (Malle, 1984). A number of investigators reported the detection of anthracene in surface water sediments. A few authors used the sediment analysis to establish the source of these compounds in the environment and the mode of this transport in aquatic environment (Windsor and Hites, 1979; Eadie et al., 1982; Tan and Heit, 1981; Sporstol et al., 1983; Boehm and Farrington, 1984). Unseparated anthracene/phenanthrene at a concentration as high as 6.4 mg/kg was reported in a sediment sample from an estuary between England and Wales (John et al., 1979).

Anthracene has been reported in groundwater from a few contaminated sites. For example, Rostad et al. (1985) reported the quantitative detection of anthracene in groundwater from a coal tar waste aquifer in St. Louis Park, MN. Bedient et al. (1984) reported the detection of up to 168.6 µg/ℓ of anthracene in groundwater from a creosote waste site in Conroe, TX.

The detection of anthracene in Canadian drinking water derived from Great Lakes was reported by Williams et al. (1982). The combined concentration of anthracene/phenanthrene (unseparable) in these waters ranged between 0.6 and 1269 ng/ℓ. The highest concentration was obtained in a water from Sault Ste. Marie collected during the summer. Anthracene has also been reported to be present in Nordic drinking water at a concentration range of 0.04-9.7 ng/ℓ (Kveseth et al., 1982). In Japanese drinking water, Shinohara et al. (1981) reported the detection of 1.7 µg/ℓ of combined anthracene and phenanthrene. Piet and Morra (1979) reported the detection of a maximum of 30 µg/ℓ of anthracene in bank-filtered tapwater in the

Netherlands. Finished waters from 13 different locations throughout the United States were analyzed for anthracene (Sorrell et al., 1980). Anthracene was not detected in any of these drinking water samples.

3.2. AIR

The sources of PAHs including anthracene in the atmosphere have been reported extensively (Santodonato et al., 1981; Daisey et al., 1986; Gammage, 1983). Some of the conventional sources are vehicular emissions, coal burning, oil burning, wood combustion, coke plants, aluminum plants, iron and steel works, foundries, ferro-alloy plants and municipal incinerators. Some of the more common modern sources of PAHs including anthracene are synfuel plants and oil shale plants. The ratios of anthracene and benzo(a)pyrene to benzo(e)pyrene and the concentrations of benzo(a)pyrene in particulate from various sources are given in Table 3-1.

The concentration of anthracene in municipal fly ash was reported to vary between 4 and 380 µg/kg (Eiceman et al., 1981). The workroom atmospheric concentration of anthracene in an aluminum reduction plant has been reported to be as high as 33.1 µg/m³ (Bjoerseth et al., 1978). Personnel monitoring of an anode plant showed that the concentration of particulate anthracene varied from none detected for pitch dust sweepers to 1.9 µg/m³ for pitch bin workers (Bjoerseth et al., 1978). The concentrations of atmospheric combined anthracene and phenanthrene inside a solvent refined coal pilot plant facility was reported to vary between 1.8 and 43.2 µg/m³ (Gammage, 1983). Personal air samples taken in the coal preparation area of the solvent refined coal pilot plant showed combined anthracene/phenanthrene concentrations of none detected to 15.7 µg/m³ (Gammage, 1983). The simulated incineration of poly(vinyl chloride) at 850°C was qualitatively shown to produce anthracene (Hawley-Fedder et al.,

TABLE 3-1

Ratios of Anthracene to Benzo(e)pyrene, Benzo(a)pyrene to Benzo(e)pyrene and the Concentrations of Benzo(a)pyrene from Various Sources of Emission*

Ratios and Concentration	Tunnel	Residential Coal-Burning	Residential Oil-Burning	Coke Oven	Auto Exhaust	Wood Combustion
Anthracene to benzo(e)pyrene ratio	2.2	0.4-7.5	0.6	0.2-6.9	6.9-17	1.1-20
Benzo(a)pyrene to benzo(e)pyrene ratio	1.2	0.6-2.0	0.9	1.0-2.3	0.3-1.4	0.5-3.0
Benzo(a)pyrene concentrations in particulate matter from various sources, ppm (mg/kg)	66-500	10-600	NR	1400-5800	NR	3-870,900

*Source: Daisey et al., 1986

NR = Not reported

1984). The concentrations of PAHs in the air of three different types of wood-heated saunas were reported by Hasanen et al. (1984). The anthracene concentrations in sauna air varied from 0.3-25.2 μg/m³.

The concentrations of anthracene in ambient air in some cities in the United States and around the world have been reported by several investigators. Table 3-2 shows the ambient atmospheric concentrations of anthracene in various locations. It is evident from Table 3-2 that the atmospheric concentrations of anthracene (even after consideration of phenanthrene contribution) measured for Osaka, Japan, in 1977-1978 are substantially higher than the other reported values. Since the authors used a small packed (6 ft.) GC column and flame ionization detecter for quantification of compounds, it is likely that complete separation of some compounds was not achieved by this analytical technique. Grosjean (1983) demonstrated that the atmospheric anthracene concentrations showed both seasonal and diurnal variations, with anthracene concentrations higher in winter and during the night. Although suitable data were not available on anthracene for comparison, Grosjean (1983) estimated that the levels of other PAHs in Los Angeles air did not significantly change during the last decade. If one assumes that the mean level of anthracene is similar to Los Angeles air (mean of 0.54 ng/m³) and that an adult individual inhales 20 m³ air/day, the average daily intake of anthracene from inhalation is 11 ng.

3.3. FOOD

The levels of anthracene in different foods have been studied much less extensively than the levels of some other PAHs, such as benzo(a)pyrene. The lack of evidence for carcinogenicity of the compound (IARC, 1983) and analytical difficulty in quantifying anthracene may have contributed to the lack of data pertaining to its level in foods. Anthracene has been reported

TABLE 3-2

Ambient Atmospheric Concentrations of Anthracene in Various Locations

Location	Year Sampled	Anthracene Concentration (ng/m^3)	Reference
Columbia, SC	1981-1982	0.3-$\geq$4.2	Keller and Bidleman, 1984
Savannah River Plant, SC	1982-1983	0.09-$\geq$0.2	Keller and Bidleman, 1984
Gainesville, FL	NR[a]	1.0	Kerkhoff et al., 1985
Jacksonville, FL	NR[a]	6.0	Kerkhoff et al., 1985
Portland, OR	1984	2.8	Ligocki et al., 1985b
Los Angeles, CA	1981-1982	0-4.82 (0.54)[b]	Grosjean, 1983
Budapest, Hungary	1971-1972	4.4-618 (61.5)[b]	Kertesz-Saringer and Morlin, 1975
Singapore	1983	<0.1	Ang et al., 1986
Osaka, Japan	1981-1982	0.17-0.57 (0.32)[b]	Matsumoto and Kashimoto, 1985
Osaka, Japan	1977-1978	52.1-294.5[c]	Yamasaki et al., 1982

[a]The year of sampling was not reported by the authors, but appears to be 1982.

[b]These are the mean concentrations.

[c]The reported concentrations were for the combined concentrations of anthracene and phenanthrene.

to be present in smoked foods, liquid smoke, charcoal-broiled steaks (Fazio and Howard, 1983) and in edible aquatic organisms collected from certain contaminated waters. The levels of anthracene in different foods are given in Table 3-3.

It is evident from Table 3-3 that data regarding the levels of anthracene in a few foods have been reported, but no data on the level of this compound in food composites used by an individual in the United States are available. Until such data are available, it is not possible to estimate the human intake of anthracene through consumption of foods.

3.4. SUMMARY

Anthracene is ubiquitous in the aquatic environment. Anthracene at a concentration range of <13-105 ng/ℓ was reported in the effluent from a sewage treatment plant in Norway (Kveseth et al., 1982). It was also reported in the concentration range of <0.03-0.84 µg/ℓ in the effluent from coal oven plants (Walters and Luthy, 1984; Griest, 1980). It has been reported to be present in several surface waters (Hites, 1979; Dewalle and Chian, 1978; Staples et al., 1985). A maximum concentration of 12.1 µg/ℓ was reported in the water from Tennessee River in Calvert City, KY (Goodley and Gordon, 1976). In general, anthracene occurs in ambient waters at a frequency of 4% and at a median concentration of <10 µg/ℓ (Staples et al., 1985). Anthracene has been reported to be present in groundwater from a few contaminated sites. Bedient et al. (1984) reported the detection of up to 168.6 µg/ℓ of anthracene in groundwater from a creosote waste site in Conroe, TX. The detection of anthracene in drinking waters throughout the world have been reported (Williams et al., 1982; Kveseth et al., 1982; Shinohara et al., 1981; Piet and Morra, 1979). The highest concentration of combined anthracene/phenanthrene at 1269 ng/ℓ was reported in

TABLE 3-3

Anthracene Levels in Different Foods

Food	Anthracene Concentration (µg/kg)	Reference
Electric broiled Japanese horse mackerel	0.2-1.9	Fazio and Howard, 1983
Gas broiled Japanese horse mackerel	2.0-2.3	Fazio and Howard, 1983
Charcoal-broiled steaks	4.5	Fazio and Howard, 1983
Barbecued ribs	7.1	Fazio and Howard, 1983
Mussel composite (U.S.) (*M. edulis* and *M. californianus*)	7.9-32*	Galloway et al., 1983
Nigerian-preserved freshwater fish	0.20-30.1	Afolabi et al., 1983
Mussell (Greece) (*M. galloprovincialis*)	8-9	Iosifidou et al., 1982
Fish (U.S.)	<20-100*	DeVault, 1985
Clams (Australia) (*Tridacma maxima*)	≤3.2	Smith et al., 1984

*These are values for combined anthracene/phenanthrene concentrations.

drinking water in Sault Ste. Marie (Williams et al., 1982). Finished waters from 13 different locations throughout the United States, however, failed to show the presence of any anthracene (Sorrell et al., 1980).

The concentrations of anthracene in ambient air in some cities in the United States and around the world have been reported. The mean concentration of anthracene in the air of Los Angeles during 1981-1982 was reported to be 0.54 ng/m³ (Grosjean, 1983), whereas its mean concentration in Osaka, Japan, during the same period was reported to be 0.32 ng/m³ (Matsumoto and Kashimoto, 1985). It has been demonstrated by Grosjean (1983) that the atmospheric anthracene concentrations show both seasonal and diurnal variations, with the anthracene concentration higher in winter and during the night. Although suitable data were not available on anthracene for comparison, Grosjean (1983) estimated that the levels of other PAHs in Los Angeles air did not change significantly during the last decade. Based on the assumptions that the mean level of anthracene in U.S. air is 0.54 ng/m³ (same as concentration in Los Angeles) and that an adult individual inhales 20 m³ air/day, the average daily intake of anthracene from inhalation is 11 ng.

Anthracene has been reported to be present in smoked foods, liquid smoke, charcoal-broiled steaks and edible aquatic organisms collected from certain contaminated waters (Fazio and Howard, 1983; Galloway et al., 1983). For example, the concentrations of anthracene in charcoal-broiled steaks and barbecued ribs were reported to be 4.5 and 7.1 µg/kg, respectively (Fazio and Howard, 1983). The U.S. National Mussel watch program collected mussels from >100 sites on the East, West and Gulf coasts (Galloway et al., 1983).

The concentration of anthracene in these mussel composites was 7.9-32 µg/kg (Galloway et al., 1983). Until data on the level of this compound in food composites used by an individual in the United States are available, it is not possible to estimate the human intake of anthracene through consumption of foods in the United States.

4. PHARMACOKINETICS

4.1. ABSORPTION

Excretion data suggest that gastrointestinal absorption of anthracene may be relatively low. As detailed in Section 4.4., 53-84% of anthracene ingested from diets containing 0.2-1.0%, and 64-74% of anthracene administered by stomach tube at 200 mg/rat were eliminated in the feces by rats in 2-3 days (Chang, 1943); urine was not analyzed and biliary excretion was not measured. The amount of anthracene in the feces was determined gravimetrically. No information was available for low-dose ingestion of anthracene.

[9-^{14}C]Anthracene (1 nmol suspended in 10% DMSO in 0.9% saline) was administered to 24 female F344/Crl rats in a single intratracheal instillation (Bond et al., 1985). Groups of 3 rats were sacrificed after 1, 3, 6, 12, 24, 48, 72 and 96 hours for determination of amount of ^{14}C remaining in the lungs. Clearance of ^{14}C from the lungs was biphasic, with 99.7 and 0.3% of the administered radioactivity cleared with half-times of 0.1 and 25.6 hours, respectively. PAHs, in general, are highly lipid soluble and are absorbed readily from the gastrointestinal tract and lungs (U.S. EPA, 1980b).

4.2. DISTRIBUTION

Pertinent data regarding the distribution of anthracene could not be located in the available literature as cited in the Appendix.

4.3. METABOLISM

Metabolites resulting from monooxygenase attack at the 1,2-bond of anthracene have been identified in the urine of rats and rabbits treated with anthracene in diet (Sims, 1964; Boyland and Burrows, 1935; Boyland and Levi, 1935) and in _in vitro_ studies that incubated anthracene with rat liver microsomes (Akhtar et al., 1979). The 1,2-dihydrodiol of anthracene appears

to be the major metabolite (Akhtar et al., 1979). Products resulting from oxidation at the 9- and 10-positions of anthracene were also identified in rat urine in the *in vivo* study (Sims, 1964), but not *in vitro* (Akhtar et al., 1979), suggesting possible nonhepatic origin; these metabolites include 9,10-dihydrodiol and 2,9,10-trihydroxyanthracene. The metabolites are excreted at least in part as glucuronide or sulfate conjugates (Sims, 1964).

4.4. EXCRETION

Groups of four male white rats were fed diets that contained 0.2, 1.0 or 1.0% anthracene in two 1-hour feedings during the same day (Chang, 1943); total intake was 270, 605 and 830 mg anthracene, respectively. Feces collected during the 2 days following administration contained 53, 82 and 84% of the ingested anthracene, respectively. Anthracene in aqueous starch suspension was administered to two male white rats by stomach tube at a dose of 200 mg (Chang, 1943). Feces collected during the 3 days after dosing contained 64 and 74% of the anthracene administered; the amount of anthracene in the feces in the above experiments, however, was determined gravimetrically and the urine was not analyzed.

Guinea pigs injected intravenously with a colloidal suspension of anthracene showed uncharacterized fluorescent materials in the bile and urine within 1 hour (Peacock, 1936). A secondary review of this study (U.S. EPA, 1981) indicated that additional information regarding this study was not available.

4.5. SUMMARY

Limited information is available regarding the pharmacokinetic profile of anthracene. Gastrointestinal absorption may be poor, as 53-84% of anthracene administered by diet or stomach tube was eliminated in the feces by rats in 2-3 days (Chang, 1943); however, urinary and biliary excretion

were not measured. Radioactivity from single intratracheal instillations of ^{14}C-anthracene was cleared from the lungs in a biphasic manner with half-times of 0.1 hours (99.7% of dose) and 25.6 hours (0.3% of dose) (Bond et al., 1985). The distribution of anthracene to tissues does not appear to have been investigated. Metabolites resulting from epoxidation at the 1,2-bond or oxidation at the 9- and 10-positions have been identified in *in vivo* and *in vitro* studies with rats (Sims, 1964; Akhtar et al., 1979). Trans-1,2-dihydroxy-1,2-dihydroanthracene and sulfate and glucuronide conjugates consistent with the formation of anthracene-1,2-oxide appear to be the major products. Orally administered anthracene appears to be eliminated by rats primarily (53-84%) in the feces (Chang, 1943). Metabolites of orally administered anthracene have been detected in the urine of rats (Sims, 1964).

5. EFFECTS

5.1. CARCINOGENICITY

A group of 28 fourteen-week-old BDI or BDIII rats of unspecified sex were given diets that initially contained 5 mg, and later (not further specified) 15 mg of "highly purified" anthracene in oil on 6 days/week for 78 weeks (Schmahl, 1955). The total dose was 4.5 g/rat and the animals were observed for life. Mean survival time was 700 days. Tumors developed in 2/28 rats and consisted of a liver sarcoma after 18 months and a uterine adenocarcinoma after 25 months. A control group was not used in this study and the tumors were not ascribed to treatment.

Single direct injections of heat-liquified beeswax-tricaprylin (1:1) mixture containing 0.5 mg anthracene into the lungs of 3- to 6-month-old female Osborne-Mendel rats did not induce keratinizing squamous metaplasia or epidermoid carcinomas at the pellet site after 4-16 weeks (8 rats), 17-29 weeks (1 rat) or 43-55 weeks (28 rats) (Stanton et al., 1972). Nonspecific granulomatous reactions occurred in all of these animals. Treatment of several groups of rats with 5-200 µg 3-methylcholanthrene under the same experimental conditions produced pulmonary epidermoid carcinomas within the first year.

Anthracene has also been tested for carcinogenicity by skin application with and without ultraviolet radiation in mice, in skin initiation-promotion assays with mice, by subcutaneous and intraperitoneal injection in rats, and by implantation into the brain or eyes in rabbits (Table 5-1). The results of the skin application studies with anthracene do not provide evidence of carcinogenicity, but contradictory results were obtained when anthracene was applied to skin together with exposure to ultraviolet radiation. Initiating activity was not indicated in the mouse skin initiation-promotion assays.

TABLE 5-1

Dermal, Injection and Implantation Carcinogenicity Assays of Anthracene

Route	Species/	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/NR	100/NR	NR	dose and number of applications not specified; 40% solution in lanolin	NR	No skin tumors; 45 and 6 mice survived >6 months and >160 days, respectively	Kennaway, 1924a,b
Skin	mouse/NR	41/NR	NR	dose and number of applications not specified; unspecified solutions in water, benzene or sesame oil	life (133 days average)	No skin tumors; dibenz[a,h]anthracene was tumorigenic in same study	Pollia, 1939
Skin	mouse/NR	50/NR	NR	dose not specified; 0.3% solution in benzene twice weekly	732 days	No skin tumors; 34 and 16 alive at 6 months and 1 year, respectively	Bachmann et al., 1937
Skin	mouse/Swiss	5/F	NR	dose not specified; 10% solution in acetone 3 times/week on the back	life (10-20 months)	No skin tumors; benzo[a]pyrene induced high incidences of skin papillomas and carcinomas under the same conditions	Wynder and Hoffmann, 1959
Skin	mouse/NR	44/NR	NR	dose not specified; 5% solution in petroleum jelly-olive oil 3 times/week on the ear	life (11 months)	No skin tumors; 1/44 alive after 11 months; primary report not available	Miescher, 1942
Skin	mouse/NR	44/NR	NR	treatment as above but with ultraviolet radiation (>320 nm) for 40 or 60 minutes, 2 hours after skin application	life (10 months)	No skin tumors; 5/44 alive after 7 months	Miescher, 1942
Skin	mouse/NR	100/NR	NR	treatment as above but mice received ultraviolet radiation for 90 minutes	life (10 months)	No skin tumors; 7/100 alive after 7 months	Miescher, 1942
Skin	mouse/NR	NR/NR	NR	dose and number of applications not specified; 10% solution in petroleum jelly-olive oil followed by unspecified exposure to ultraviolet (320-405 nm) alone or with visible light	NR	"High incidence" of skin tumors, including many carcinomas, was observed after 5-8 weeks; no tumors in controls treated with anthracene, ultraviolet or ultraviolet with visible light; primary report not available; unusually short latency and inadequate histopathology reporting noted by IARC (1983)	Heller, 1950
Skin	mouse/Skh: hairless-1	24/mixed	NR	0 or 4 µg in methanol once daily, followed by ultraviolet (>290 nm) for 2 hours, 5 days/week	38 weeks	Incidence of skin tumors not significantly increased in treated group; survival was 20/24 and 19/24 in controls and treated groups	Forbes et al., 1976

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TABLE 5-1 (cont.)

Route	Species/	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/S	20/NR	NR	two applications (0.3 mℓ of 0.5% solution in acetone per application) with a 30-minute interval 3 times/week for a total of 20 applications (30 mg/animal total); 18 weekly applications of croton oil in acetone (0.3 mℓ) consisting of 1/0.17%, 2/0.085% and 15/0.17% croton oil, beginning 25 days after the first anthracene application; controls received the same treatment with croton oil only.	25 weeks	Incidence of skin tumors not significantly increased in treated groups; survival was 19/20 and 17/20 in the control and treated groups, respectively	Salaman and Roe, 1956
Skin	mouse/CD-1	30/F	chromatographically purified	single application of 10 µmol (1782 µg) in benzene followed 1 week later by 5 µmol TPA twice weekly for 34 weeks; controls received the same treatment with TPA only	35 weeks	Incidence of skin papillomas was 4/28 (14%) in treated and 1/30 (3%) in controls	Scribner, 1973
s.c.	rat/NR	10/NR	NR	weekly injections of 2 mℓ of 0.05% suspension in water for life (103 mg/animal maximum total dose)	>18 months	No subcutaneous sarcomas; survival was 7/10 after 12 months and 8/10 after 18 months; dibenz[a,h]-anthracene was tumorigenic under the same conditions	Boyland and Burrows, 1935
s.c.	rat/Wistar	5/NR	NR	5 mg in sesame oil, once weekly for 6 or 7 weeks	10 months	No tumors; examinations apparently included viscera; dibenz[a,h]-anthracene induced subcutaneous tumors in 2/5 similarly treated rats	Pollia, 1941
s.c.	rat/BDI and BDIII	10/NR	highly purified	20 mg in unspecified oil once weekly for 33 weeks	life	Injection site tumors (fibromas with sarcomatous areas) in 5/9 rats, mean latency, 26 months; no controls, but rats treated similarly with naphthalene in oil did not develop local tumors	Schmahl, 1955
i.p.	rat/BDI or BDIII	10/NR	highly purified	20 mg in unspecified oil once weekly for 33 weeks	life (mean ~2 years)	Tumor in one rat (spindle-cell sarcoma in abdominal cavity); no control group	Schmahl, 1955

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TABLE 5-1 (cont.)

Route	Species/	No.*/Sex	Purity	Treatment	Duration	Effects/Comments	Reference
Cerebral cortex implant	rabbit/NR	5/NR	NR	10 mg (1 rabbit) or 20 mg (4 rabbits) pellets	4.5 years	No tumors; survival was 4/5 after 4 years and 2/5 at 4.5 years	Russell, 1947
Cerebellar implant	rabbit/NR	2/NR	NR	12 mg pellets	4.5 years	No tumors; survival was 2/2 at 4.3 years	Russell, 1947
Optic implant	rabbit/NR	2/NR	NR	4 or 5 mg pellets	4.5 years	No tumors; survival was 2/2 at 4.5 years	Russell, 1947

*Numbers in treatment and control (if used) group(s) unless specified otherwise.

NR = Not reported; s.c. = subcutaneous; i.p. = intraperitoneal

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The injection and implantation studies do not provide evidence of carcinogenicity, but these results cannot be regarded as conclusive because of inadequacies in experimental design (e.g., small numbers of animals, limited number of exposures, inadequate controls).

Three cases of epithelioma (hand, cheek and wrist) were reported in men engaged in handling 40% crude anthracene in an alizarin factory (Kennaway, 1924a,b). Two of these workers had worked for 30 and 32 years with the crude anthracene and had never worked with any other tar product. Workers in the same factory who had contact with only purified anthracene did not develop tumors or other skin lesions (acne, keratoses, telangiectases, pigmentation) that affected those who had contact with the crude material. The crude anthracene was not characterized and additional information regarding these observations was not reported.

5.2. MUTAGENICITY

Anthracene has been tested in numerous mutagenicity and other short-term assays with negative results. IARC (1983) has reviewed many of these studies, which include such assays with prokaryotes as DNA damage/repair in Escherichia coli and Bacillus subtilis, reverse mutation in Salmonella typhimurium including strains TA1535, TA1537, TA1538, TA98 and TA100; various assays in fungi including mitotic recombination in Saccharomyces cerevisiae; assays in mammalian cells in vitro, unscheduled DNA synthesis in primary rat hepatocytes and HeLa cells, mutation in Chinese hamster V79 and mouse lymphoma L5178Y cells, sister chromatid exchange and/or chromosome breaks in Chinese hamster D6 or rat liver epithelial cells, cell transformation in mouse BALB/3T3, guinea-pig fetal or Syrian hamster embryo cells; and mammalian cells in vivo, sister chromatid exchange and chromosome aberrations in Chinese hamsters, micronuclei in mice. Anthracene also produced

negative responses in mutagenicity and short-term assays that were reported in the more recent literature, including induction of prophage (inductest) (Mamber et al., 1984) and sfiA gene (SOS Chromotest) (Quillardet et al., 1985) in E. coli, mutation at the HGPRT locus and sister chromatid exchanges in rat liver epithelial cells in vitro (Ved Brat et al., 1983), mutation to 6-thioguanine resistance in Chinese hamster V79 cells in vitro (Langenbach et al., 1983), DNA single strand breaks in rat hepatocytes in vitro (Sina et al., 1983) and neoplastic transformation in C3H/10T1/2 Clone 8 cells (Lubet et al., 1983). Exogenous metabolic activation systems were used in most of the aforementioned in vitro assays. Many of the assays were included in validation studies, where anthracene was presumed to be noncarcinogenic.

Although most of the available data indicate unequivocal nonmutagenicity of anthracene, weak mutagenicity was recently demonstrated in the Ames assay with a new tester strain (TA97) of S. typhimurium (Sakai et al., 1985). Mutagenic activity was expressed only in the presence of S-9 metabolic activation preparation, and was not observed with strains TA98 or TA100 with or without metabolic activation.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of anthracene could not be located in the available literature as cited in the Appendix.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of anthracene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding toxic effects of chronic or subchronic oral or inhalation exposure to anthracene could not be located in the available

literature as cited in the Appendix. Information regarding nonneoplastic effects was not reported in the only oral carcinogenicity study of anthracene (Schmahl, 1955) (see Section 5.1.).

Holland et al. (1980) administered uncharacterized oil shale containing polycyclic aromatic hydrocarbons, including anthracene, to Syrian golden hamsters by inhalation of 50 mg respirable shale dust/m^3 for 4 hours/day, 4 days/week. The authors reported interim results indicating that shale dust caused little pulmonary epithelial or fibrotic reaction, but that retorted shales caused inflammation accompanied by fibrosis. Because of the uncharacterized nature of the test material, it is not possible to quantify these or future data from this study for use in risk assessment.

5.6. OTHER RELEVANT INFORMATION

The abstract of a Russian study indicated that single oral doses of 1.47 or 2.44 g/kg of commercial grade anthracene or 17 g/kg of pure anthracene were not lethal to mice (Nagornyi, 1969). Toxic effects reportedly included fatigability, adynamia, histological hyperemia in the kidney, liver, heart and lungs, "lipid dystrophy" in the liver and leukocytosis with neutrophilia.

Daily intragastric administration of 100 mg/kg anthracene in olive oil for 4 days produced a small (3-fold) increase in mean liver cytosolic aldehyde dehydrogenase activity in a group of five male Wistar rats (Torronen et al., 1981). Treatment-related effects on aldehyde dehydrogenase activity in the liver microsomes or postmitochondrial fractions of small intestinal mucosa or effects on liver/body weight ratios were not observed.

Salamone et al. (1981) reported that the single-dose intraperitoneal LD_{50} for mice is >430 mg/kg. Gerarde (1960) found that 9/10 mice survived daily intraperitoneal injections of 500 mg/kg anthracene for 7 days with depressed weight gain.

Systemically administered anthracene (50 mg/mL corn oil by gavage) with ultraviolet irradiation of the skin for 1 hour, 2 hours after dosing, produced keratitis of the exposed skin in mice (Dayhaw-Barker et al., 1985). This effect reportedly was less pronounced in mice exposed only to ultraviolet light alone and not evident in vehicle controls. Topically applied anthracene increases the sensitivity of human and hairless mouse skin to ultraviolet light (IARC, 1983; U.S. EPA, 1981; Kaidbey and Nonaka, 1984).

5.7. SUMMARY

Administration of diets that supplied a total dose of 4.5 g/rat of anthracene over 78 weeks produced tumors in 2/28 rats (a liver sarcoma and a uterine adenocarcinoma) that were observed for life (Schmahl, 1955). A control group was not used and the tumors were not ascribed to treatment. A single intrapulmonary injection of 0.5 mg anthracene in beeswax-tricaprylin mixture did not induce local neoplastic responses in rats after 4-55 weeks of observation (Stanton et al., 1972).

Twice or thrice weekly skin applications of anthracene for life did not produce local tumors in mice (Bachmann et al., 1937; Wynder and Hoffmann, 1959; Miescher, 1942), but contradictory results were obtained when anthracene was applied to mouse skin with concurrent or directly subsequent ultraviolet irradiation (Heller, 1950; Forbes et al., 1976). Mouse skin initiation-promotion assays using croton oil (Salaman and Roe, 1956) or TPA (Scribner, 1973) as the promoter did not indicate a tumor initiating effect of anthracene. Weekly subcutaneous injections of anthracene in rats for 6 weeks to life (Pollia, 1941; Schmahl, 1955; Boyland and Burrows, 1935), weekly intraperitoneal injections in rats for 33 weeks (Schmahl, 1955) or brain or eye implants in rabbits for 4.5 years (Russell, 1947) did not produce local tumors, but these findings should be regarded as inconclusive because of inadequacies in experimental design.

Although Sakai et al. (1985) recently obtained a weak mutagenic response with activation for anthracene using a new tester strain (TA97) of S. typhimurium, this represents the only positive response. Anthracene has been tested in numerous mutagenicity and other short-term assays with negative results (IARC, 1983; Langenbach et al., 1983; Lubet et al., 1983; Ved Brat et al., 1983; Mamber et al., 1984; Quillardet et al., 1985). These include DNA damage, mutation, cytogenicity and transformation assays with bacteria, yeast and mammalian cells in vitro and in vivo. Exogenous metabolic activation systems were used in most of the in vitro assays.

Pertinent data regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of anthracene could not be located in the available literature as cited in the Appendix.

6. AQUATIC TOXICITY

6.1. ACUTE

The available information concerning acute toxicity of anthracene to aquatic animals is presented in Table 6-1. The most sensitive species tested was Daphnia pulex, with a 15-minute EC_{50} for immobilization value of 1.2 µg/ℓ in natural sunlight (Allred and Giesy, 1985; Oris et al., 1984).

The toxicity of anthracene is greatly influenced by lighting conditions. Anthracene is essentially nontoxic within solubility limits under normal laboratory fluorescent lighting (wavelengths >380 nm) (Allred and Giesy, 1985). Several studies, however, have shown that the toxicity of anthracene is substantially increased under natural lighting conditions (Allred and Giesy, 1985; Bowling et al., 1983; Kagan et al., 1985; Oris et al., 1984). Allred and Giesy (1985) found that anthracene was not toxic to Daphnia pulex at 30 µg/ℓ under fluorescent lighting, but caused 100% immobilization in 2 minutes under natural sunlight. The available information indicates that this photo-enhanced toxicity is due to material associated with the organisms themselves rather than anthracene in the water. Allred and Giesy (1985) found that Daphnia pulex that were exposed to anthracene, then transferred to clean water and then exposed to sunlight still experienced the toxic effects (immobilization). Bowling et al. (1983) demonstrated the same phenomenen with bluegills (Lepomis macrochirus). Fish exposed to 12.7 µg/ℓ in sunlight were killed; however, fish held in the shade downstream from a sunlit area at this concentration did not die. When transferred to clean water and sunlight, previously exposed fish died, indicating again that anthracene accumulated by the animals was the cause of the photo-enhanced toxicity. The influence of lighting conditions on anthracene toxicity may explain why some of the acute toxicity data are quite variable.

TABLE 6-1

Acute Toxicity of Anthracene to Aquatic Animals

Species	Concentration (µg/L)	Effect	Reference
FISH			
Fathead minnow, Pimephales promelas	360	24-hour LC_{50}	Kagan et al., 1985
Bluegill, Lepomis macrochirus	11.9	96-hour LC_{50}	Oris et al., 1984
	12.7	mortality	Bowling et al., 1983
AMPHIBIANS			
Frog, Rana pipiens	110	24-hour LC_{50}	Kagan et al., 1985
	25	5-hour LC_{50}	Kagan et al., 1984
INVERTEBRATES			
Water flea, Daphnia magna	20	24-hour LC_{50}	Kagan et al., 1985
	3030	48-hour LC_{50}	Bobra et al., 1983
Water flea, Daphnia pulex	1.2	15 minute LC_{50}, immobilization, in natural sunlight	Allred and Giesey, 1985; Oris et al., 1984
	9.6	100% immobilization, 10 minutes, in natural sunlight	Allred and Giesy, 1985
Mosquito, Aedes aegypti	150	24-hour LC_{50}	Kagan et al., 1985
Mosquito, Culex sp.	26.8	24-hour LC_{50}	Oris et al., 1984
Brine shrimp, Artemia salina	20	24-hour LC_{50}	Kagan et al., 1985
Mussel, Mytilus edulis	50-200	decreased lysosomal stability, digestive gland	Moore and Ferrar, 1985
PROTOZOA			
Ciliate, Paramecium caudatum	1000	90% lethal, 60 minutes	Epstein et al., 1963

For example, LC_{50} values of 20 and 3030 µg/ℓ were reported for Daphnia magna (Bobra et al., 1983; Kagan et al., 1985).

6.2. CHRONIC

Pertinent data regarding chronic toxicity of anthracene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

There is little information regarding the effects of anthracene on aquatic plants. Hutchinson et al. (1980) reported EC_{50} values of 239 and 535 µg/ℓ for inhibition of photosynthesis in Chlamydomonas angulosa and Chlorella vulgaris, respectively. Giddings (1979) found that a 100% saturated anthracene solution had no effect on photosynthesis in Selenastrum capricornutum.

Anthracene toxicity to aquatic plants also seems to be influenced by lighting conditions. Cody et al. (1984) stated that growth of Selenastrum capricornutum was unaffected by 40 mg/ℓ under gold fluorescent light, but was inhibited 30% at 8 mg/ℓ under cool-white fluorescent light.

6.4. RESIDUES

Data from bioconcentration experiments with anthracene are presented in Table 6-2. The largest BCF was 16,800 for the benthic amphipod, Pontoporcia hoyi (Landrum, 1982). The ability to metabolize and eliminate anthracene seems to vary considerably among different species. The fish that have been tested (rainbow trout, dolly varden char and bluegill) do not appear to metabolize anthracene rapidly (Linder and Bergman, 1984; Spacie et al., 1983; Thomas and Rice, 1982). Some invertebrates such as oysters, Crassostrea virginica (Lee et al., 1978), and the amphipod, Pontoporlia hoyi, (Landrum, 1982) also metabolize and eliminate anthracene slowly, but other species such as the midge, Chironomus viparius, metabolize and eliminate

TABLE 6-2

Bioconcentration Data for Anthracene in Aquatic Organisms

Species	Concentration (µg/l)	BCF	Remarks	Reference
FISH				
Rainbow trout, *Salmo gairdneri*	36	200	18 hours	Linder and Bergman, 1984
	50	779	72 hours	Linder et al., 1985
	NR	9000-9200	estimated steady-state BCF	Linder et al., 1985
Goldfish, *Carassius auratus*	NR	162	NC	Ogata et al., 1984
Bluegill, *Lepomis macrochirus*	0.7-16.6	675	estimated steady-state BCF based on anthracene only	Spacie et al., 1983
	0.7-16.6	900	estimated steady-state BCF based on total ^{14}C accumulation	Spacie et al., 1983
CRUSTACEANS				
Water flea, *Daphnia magna*	NR	200	1-hour BCF	Herbes, 1976
	30-38	450-500	20-hour BCF, no particulates in water	McCarthy, 1983
	30-38	230-370	20-hour BCF in the presence of particulates	McCarthy, 1983
Water flea, *Daphnia pulex*	0.02	760	24-hour, steady-state BCF	Herbes and Risi, 1978
	NR	917	24-hour BCF	Southworth et al., 1978
Amphipod, *Hyalella azteca*	NR	1800	BCF corrected for biotransformation	Landrum and Scavia, 1983
Amphipod, *Pontoporeia hoyi*	NR	16,800	estimated steady-state BCF	Landrum, 1982
INSECTS				
Mayfly, *Hexagenia* sp.	NR	3500	24-hour BCF	Herbes, 1976
Midge, *Chironomus riparius*	1.7-30.5	1702-1964	30-hour BCF, based on ^{14}C accumulation	Gerould et al., 1983
	1.7-30.5	47-132	4-hour BCF, based on anthracene only	Gerould et al., 1983
MOLLUSCS				
Clam, *Tapes japonica*	0.04-0.21	800-1300	1- to 7-day BCF	Obana et al., 1983
Oyster, *Crassostrea virginica*	13	431	2-day BCF	Lee et al., 1978
	1	2500	8-day BCF	
PLANTS				
Green alga, *Chlorella fusca*	50	7800	24-hour BCF	Geyer et al., 1981
	NR	7770	24-hour BCF	Geyer et al., 1984

NR = Not reported; NC = no comment

anthracene rapidly (Gerould et al., 1983). The presence or absence of sediment in these studies may also influence the calculated BCFs because sediment can be a major source of anthracene uptake, especially for benthic organisms such as amphipods (Eadie et al., 1983; Landrum and Scavia, 1983).

There are relatively little residue monitoring data for anthracene compared with other polycyclic aromatic hydrocarbons. Tsuji et al. (1985) reported that clams (unspecified species) from Japanese waters contained 0.14-0.64 ng/g. Malins et al. (1985) found that stomach contents of English sole, Parophyrys vetulus, from Puget Sound, Washington, contained 13-460 ng/g. Maccubbin et al. (1985) reported that stomach contents of white suckers, Catostomus commersoni, from eastern Lake Erie contained 1.97-2.17 ng/g.

6.5. SUMMARY

There is relatively little information concerning the toxicity of anthracene to aquatic organisms. Acutely toxic concentrations range from 1.9 µg/ℓ for Daphnia pulex (Allred and Giesy, 1985; Oris et al., 1984) to 3030 µg/ℓ for Daphnia magna (Bobra et al., 1983). Some of this variability may be explained by the fact that anthracene toxicity is affected by lighting conditions, with toxicity increased under natural sunlight and ultraviolet radiation rather than fluorescent lights (Allred and Giesy, 1985; Bowling et al., 1983; Kagan et al., 1985). Reported toxic concentrations for aquatic plants were also highly variable. Hutchinson et al. (1980) reported that photosynthesis was inhibited in Chlamydomonas angulosa at 239 µg/ℓ, while Giddings (1979) found that Selenastrum capricornutum was unaffected by a 100% saturated solution. Reported BCF values ranged from 47-132 for Chironomus riparius (Gerould et al., 1983) to 16,800 for Pontoporlia hoyi (Landrum, 1982). These species also represent the extremes in the ability to metabolize and eliminate anthracene.

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7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Exposure criteria and TLVs have been developed for polycyclic aromatic hydrocarbons as a class, as well as for several individual polycyclic aromatic hydrocarbons. The OSHA has set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo[a]pyrene, phenanthrene, acridine, chrysene, pyrene) (OSHA, 1985). NIOSH (1977) recommended a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances of 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommended a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

Environmental quality criteria, which specify concentration limits intended to protect humans against adverse health effects, have been recommended for polycyclic aromatic hydrocarbons in ambient water. The U.S. EPA (1980b) has recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic polycyclic aromatic hydrocarbons in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo[a]pyrene, and acknowledges the conservative assumption that all carcinogenic polycyclic aromatic hydrocarbons are equal in potency to benzo[a]pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carcinogenic polycyclic aromatic hydrocarbons over an entire lifetime is estimated to keep the lifetime risk of cancer development below one chance in 100,000. Anthracene was not among the PAH considered as carcinogenic by the U.S. EPA (1980).

The EPA has not recommended an ambient water quality criterion for noncarcinogenic polycyclic aromatic hydrocarbons as a class. The U.S. EPA (1980b) acknowledged that data suitable for quantitative risk assessment of noncarcinogenic polycyclic aromatic hydrocarbons are essentially nonexistent.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of anthracene in particular could not be located in the available literature as cited in the Appendix. U.S. EPA (1980b) reported, however, that acute toxicity to saltwater aquatic life occurred at concentrations as low as 300 µg/ℓ of polycyclic aromatic hydrocarbons in general, and would occur at lower concentrations in species more sensitive than those tested. U.S. EPA (1980b) concluded that the available data at that time were inadequate to recommend criteria or make generalizations about chronic toxicity or acute toxicity to freshwater organisms.

B. RISK ASSESSMENT

Administration of diets that initially contained 5 mg and later (not otherwise specified) 15 mg of anthracene on 6 days/week for 78 weeks (total dose of 4.5 g/rat) produced tumors in 2/28 rats (a liver sarcoma and a uterine adenocarcinoma) that were observed for life (Schmahl, 1955). A control group was not used and the tumors were not ascribed to treatment. A single intrapulmonary injection of 0.5 mg anthracene in beeswax-tricaprylin mixture did not induce local neoplastic responses in rats after 4-55 weeks of observation (Stanton et al., 1972).

Twice or thrice weekly skin applications of anthracene for life did not produce local tumors in mice (Bachmann et al., 1937; Wynder and Hoffmann, 1959; Miescher, 1942), but contradictory results were obtained when anthracene was applied to mouse skin with concurrent or directly subsequent ultraviolet irradiation (Heller, 1950; Forbes et al., 1976) (see Table 5-1). Mouse skin initiation-promotion assays using croton oil (Salaman and Roe, 1956) or TPA (Scribner, 1973) as the promoter do not indicate a tumor initiating effect of anthracene. Weekly subcutaneous injections of anthracene in rats for 6 weeks to life (Pollia, 1941; Schmahl, 1955; Boyland and Burrows, 1935), weekly intraperitoneal injections in rats for 33 weeks (Schmahl, 1955) or brain or eye implants in rabbits for 4.5 years (Russell, 1947) did not produce local tumors, but these findings should be regarded as inconclusive because of inadequacies in experimental design.

Anthracene has been tested in numerous mutagenicity and other short-term assays with primarily negative results (IARC, 1983; Langenbach et al., 1983; Lubet et al., 1983; Ved Brat et al., 1983; Mamber et al., 1984; Quillardet

et al., 1985). These include DNA damage, mutation, cytogenicity and transformation assays with bacteria, yeast and mammalian cells *in vitro* and *in vivo*. Exogenous metabolic activation systems were used in most of the *in vitro* assays.

Pertinent data regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of anthracene could not be located in the available literature as cited in the Appendix.

The available information provides no evidence that anthracene is carcinogenic, therefore anthracene is a Group D chemical according to the U.S. EPA weight of evidence system (see Section 9.2.). Calculation of an RfD (formerly ADI) for anthracene is precluded by the lack of relevant toxicity data.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Pertinent data regarding chronic or subchronic toxic effects, teratogenicity or other reproductive effects of anthracene could not be located in the available literature as cited in the Appendix.

Holland et al. (1980) administered uncharacterized oil shale containing polycyclic aromatic hydrocarbons, including anthracene, to Syrian golden hamsters by inhalation of 50 mg respirable shale dust/m³ for 4 hours/day, 4 days/week. The authors reported interim results indicating that shale dust caused little pulmonary epithelial or fibrotic reaction, but that retorted shales caused inflammation accompanied by fibrosis. Because of the uncharacterized nature of the test material in this study, it is not possible to quantify those data for use in deriving an RQ (Table 9-1) to reflect hazard associated with exposure to anthracene (U.S. EPA, 1984).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Administration of diets that initially contained 5 mg and later (not otherwise specified) 15 mg of anthracene on 6 days/week for 78 weeks (total dose of 4.5 g/rat) produced tumors in 2/28 rats (a liver sarcoma and a uterus adenocarcinoma) that were observed for life (Schmahl, 1955). A control group was not used and the tumors were not ascribed to treatment. A single intrapulmonary injection of 0.5 mg anthracene in beeswax-tricaprylin mixture did not induce local neoplastic responses in rats after 4-55 weeks of observation (Stanton et al., 1972).

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TABLE 9-1

Anthracene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	
Dose:	
Effect:	
Reference:	
RV_d:	
RV_e:	
Composite Score:	
RQ:	Data are not sufficient for deriving an RQ

initiation-promotion assays using croton oil (Salaman and Roe, 1956) or TPA (Scribner, 1973) as the promoter do not indicate a tumor initiating effect of anthracene. Weekly subcutaneous injections of anthracene in rats for 6 weeks to life (Pollia, 1941; Schmahl, 1955; Boyland and Burrows, 1935), weekly intraperitoneal injections in rats for 33 weeks (Schmahl, 1955) or brain or eye implants in rabbits for 4.5 years (Russell, 1947) did not produce local tumors, but these findings should be regarded as inconclusive because of inadequacies in experimental design.

Anthracene has been tested in numerous mutagenicity and other short-term assays with primarily negative results (IARC, 1983; Langenbach et al., 1983; Lubet et al., 1983; Ved Brat et al., 1983; Mamber et al., 1984; Quillardet et al., 1985). These include DNA damage, mutation, cytogenicity and transformation assays with bacteria, yeast and mammalian cells in vitro and in vivo. Exogenous metabolic activation systems were used in most of the in vitro assays.

The available information provides no evidence that anthracene is carcinogenic.

IARC (1983) reported that there was insufficient evidence regarding the carcinogenic risk to humans and experimental animals associated with oral or inhalation exposure to anthracene. According to U.S. EPA (1986b) guidelines, there is no data for humans and inadequate evidence for animals. Applying the criteria for evaluation of the overall weight of evidence for the carcinogenic potential for humans of the Carcinogen Assessment Group of the U.S. EPA (1986b), anthracene is most appropriately designated a Group D - Not Classified chemical. Direct hazard ranking of anthracene under CERCLA is therefore precluded.

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

ACGIH (American Conference of Governmental Industrial Hygienists). 1985-1986. TLVs: Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environment with Intended Changes for 1985-1986. Cincinnati, OH. 114 p.

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Grayson, M. and D. Eckroth, Ed. 1978-1983. Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed. John Wiley and Sons, NY. 23 Volumes.

Hamilton, A. and H.L. Hardy. 1974. Industrial Toxicology, 3rd ed. Publishing Sciences Group, Inc., Littleton, MA. 575 p.

IARC (International Agency for Research on Cancer). IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. WHO, IARC, Lyons, France.

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USITC (U.S. International Trade Commission). 1983. Synthetic Organic Chemicals. U.S. Production and Sales, 1982, USITC Publ. 1422, Washington, DC.

Verschueren, K. 1983. Handbook of Environmental Data on Organic Chemicals, 2nd ed. Van Nostrand Reinhold Co., NY.

Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

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